PostScript

LETTERS

Refutation of ''the myth of the female athlete triad''

In its June 2006 issue, the *British Journal of Sports Medicine* published an article entitled ''The myth of the female athlete triad'',[1] which makes six allegations: (1) the 1997 American College of Sports Medicine (ACSM) Position Stand on the Female Athlete Triad discourages girls and women from participating in physical activity; (2) the ACSM Position Stand portrays female athletes as psychopathological; (3) no data link disordered eating to the induction of low energy availability in athletes; (4) no prospective data have shown low energy availability to cause infertility and osteoporosis in any species; (5) experiments in which low energy availability disrupted luteinizing hormone pulsatility and bone turnover in women do not apply to athletes; and (6) the female athlete triad does not exist.

The first allegation is blatantly and flagrantly false. DiPietro and Stachenfeld[1] write that ''the female athlete triad stands in contrast to other ACSM, as well as World Health Organization and US Public Health Services Guidelines, which state that participation in sport or in physical activity promotes the health and safety of girls and young women.'' In fact, the very first two sentences of the 1997 ACSM Position Stand on the Female Athlete Triad[2] are ''The majority of girls and women derive significant health benefits from regular physical activity without incurring health risks. They should be encouraged to be physically active at all phases of their lives.''

The ACSM position stand goes on to warn female athletes against the hazards of undernutrition. The ACSM has a responsible tradition of warning the public against exercising in an unhealthy manner, including positions stands warning wrestlers against the hazards of unhealthy weight loss,[3] warning runners against the hazards of heat and cold,[4] and warning all athletes against the hazards of dehydration.[5] No one interprets those position stands as discouraging physical activity, and DiPietro and Stachenfeld are alone in misrepresenting the ACSM Position Stand on the Female Athlete Triad in this way.

DiPietro and Stachenfeld's second allegation is also false. In citing disordered eating as the cornerstone of the female athlete triad in the ACSM Position Stand, they write: ''A distorted body image, disordered—for example, restrictive—eating and underweight, in essence have been implicated as the primary factors in the aetiology of the female athlete triad.'' They go on to claim that eating disorders, psychiatric illness and psychopathology are also purported to be essential features of the triad. In fact, in describing disordered eating in athletes, the authors of the ACSM Position Stand were careful to specify that, in athletes, restrictive eating behaviours include ''inadvertently failing to balance energy expenditures with adequate energy intake''. Thus, neither psychopathology nor reduced dietary energy intake has ever been a necessary precondition of the triad.

British investigators have been prominent in showing that ''there is no strong biological imperative to match energy intake to activity-induced energy expenditure''[6]: food deprivation increases hunger, but the same energy deficit produced by exercise energy expenditure does not,[7] and ad libitum energy deficiency is even more extreme when high volumes of exercise are performed on a high-carbohydrate diet, such as that recommended for athletes, than on a high-fat diet.[8] For athletes, appetite is not a reliable indicator of energy requirements. Athletes must learn to eat by discipline instead of by appetite.

DiPietro and Stachenfeld's third allegation is also false. After misrepresenting the definition of restrictive eating in the ACSM Position Stand's description of disordered eating in athletes, DiPietro and Stachenfeld execute a semantic sleight-of-hand by substituting their psychopathological definition of disordered eating into their allegation that no data link such pathological disordered eating to low energy availability in athletes. As explained above, the ACSM Position Stand requires no such linkage. In regulating physiological systems, the brain does not care why energy availability is low. It does not care whether an athlete's dietary energy intake is low, whether her exercise energy expenditure is high, whether she is trying to lose weight, whether she is pursuing a rational plan or a psychotic obsession. All it cares about is that her energy availability is low.

DiPietro and Stachenfeld's fourth allegation is also false. They write: ''Neither are there prospective data on the long term negative consequences of lower energy availability (eg infertility, osteoporosis), although its relation to longevity in several non-human species has been documented repeatedly.'' Indeed, such data are unlikely to ever be generated in human studies: after 40% restrictions consistently caused both infertility[9–11] and skeletal demineralisation[12–16] in animals, ongoing prolonged clinical trials of energy restriction in healthy humans and animals[17] limit energy restriction to 20–30%. Our experiments have shown that luteinizing hormone pulsatility is disrupted in exercising women only when their energy availability is reduced by more than about 33%.[18]

DiPietro and Stachenfeld offer athletes false comfort in the early evolution of the body's trade-off of health for survival in times of famine, because absence of psychopathology provides athletes no protection from the physical pathologies caused by low energy availability. Fracture risk doubles for every reduction of one standard deviation in bone mineral density,[19] and a mere 10% reduction accounts for >50% of differences in hip fracture rates later in life.[20] By comparison, as the number of missed menstrual cycles between menarche and age 19 years increases beyond 40, bone mineral density declines by 30%.[21] Therefore, stress fractures occur more commonly in athletes with amenorrhoea than in those with eumenorrhoea.[22–29]

DiPietro and Stachenfeld's fifth allegation is both false and hypocritical in light of their own extrapolation to athletes from *Caenorhabditis elegans*. DiPietro and Stachenfeld present three arguments in support of this allegation. Firstly, they argue that the energy deficits that disrupted luteinising hormone pulsatility within 5 days in our randomised, prospective, controlled experiments on young, habitually sedentary, regularly menstruating, exercising women[18 30 31] are unlike the gradually imposed energy deficits more likely to occur in athletic training. This argument betrays a failure to understand either the motivation for or the contribution made by our experiments, because Bullen *et al*[32] had overcome DiPietro and Stachenfeld's first argument by inducing menstrual disorders in untrained women by progressive exercise training 5 years before our experiments began. What had been left unresolved by Bullen *et al* was the mechanism by which exercise training disrupted reproductive function, as they had induced menstrual disorders in a group of subjects who were fed to maintain body weight, as well as in a second group who were fed to lose one pound of body weight per week. This suggested that stress inherent in exercise training, independent of its energy cost, might disrupt the function of the female reproductive system. If that were true, then policies to limit female participation in sports were bound to be proposed. However, we noted that weight is a poor indicator of energy sufficiency, because the suppression of physiological systems during energy deficiency reduces energy expenditure and prevents weight loss,[33] and that amenorrhoeic athletes typically report stable body weights.[26 33–39] This suggested that the menstrual disorders induced in the weight-maintenance group might have been caused by unintentional energy deficiency. If that were true, then policies to promote better nutrition would be warranted instead of policies to limit participation.

By the time we began our experiments, ovarian function had been found to depend critically on luteinizing hormone pulsatility,[40] and we had found that luteinizing hormone pulsatility and ovarian follicular development are suppressed in athletes with amenorrhoea.[41] Therefore, we proceeded to determine the independent effects of energy availability (defined as dietary energy intake minus exercise energy expenditure) and exercise stress (defined independently as everything associated with exercise, except its energy cost) on luteinizing hormone pulsatility in the follicular phase of the menstrual cycle. We disrupted luteinizing hormone pulsatility during the follicular phase of the menstrual cycle in exercising women and prevented this disruption by increasing energy intake in exact compensation for exercise energy expenditure, thereby showing that exercise has no suppressive effect on luteinizing hormone pulsatility beyond the subtraction of its energy cost from energy intake.[40] Subsequently, Williams *et al*[42 43]. confirmed that short-term effects of low energy availability on luteinizing hormone pulsatility in the follicular phase predict chronic effects on ovarian function. They induced amenorrhoea

by gradually increasing energy deficits over many months in voluntarily exercising monkeys by gradually increasing exercise training volume without reducing dietary intake,[42] and then restored ovulation by gradually increasing dietary intake without moderating the exercise regimen.[43] Thus, the ability of progressive exercise training to induce menstrual disorders, an outcome variable requiring months of observation, was shown and confirmed by other investigators. By studying the endocrine signal regulating ovarian follicular development, an outcome variable requiring only a few days of observation, we identified the specific behavioural factor in exercise training that disrupts ovarian function, and thereby discouraged policies that limit female participation in sports and encouraged policies that promote better nutrition, such as the ACSM Position Stand.

DiPietro and Stachenfeld's second argument in support of their fifth allegation is that our subjects did not have the training-related adaptations in substrate storage and conversion seen in athletes. In fact, these adaptations only occur in female athletes when they eat as much as male athletes,[44 45] but most female athletes do not.[46] On average, female athletes in most sports eat about 30% less per kilogram of body weight than male athletes in the same sports,[46] which means half of them eat even less than that. Why female athletes eat so much less than male athletes is an open question. Perhaps exercise training suppresses appetite more in women than in men. Perhaps female athletes aspire to the leaner body composition of male athletes. Perhaps motivation varies from sport to sport. As stated above, the brain does not care why they do it, but sports governing bodies might make wiser policies if they took this behaviour into consideration.

DiPietro and Stachenfeld's third argument is that the primary method of lowering energy availability in our experiments has been to restrict energy intake rather than to increase energy expenditure, as would occur with training. In fact, we have induced the low T_3 syndrome seen in amenorrhoeic athletes[47] and repeatedly disrupted luteinizing hormone pulsatility,[48 49] and others have induced luteal deficiency, anovulation and amenorrhoea[32 42] by increasing exercise energy expenditure without any reduction in dietary energy intake.

DiPietro and Stachenfeld do not seem to understand that athletes would be inappropriate subjects in experiments investigating whether low energy availability disrupts reproductive function, because the incidence of subclinical menstrual disorders in even regularly menstruating, recreational runners is about 80%,[50] and such pre-existing menstrual disorders will bias experimental results. However, amenorrhoeic athletes are appropriate subjects for investigating interventions to restore reproductive function, and the applicability of our experiments to athletes is further confirmed by the finding that menses were restored in amenorrhoeic athletes by gradually increasing energy availability to the same threshold of energy availability (about 30 kcal/kg of fat-free mass per day),[51] below which luteinizing hormone pulsatility was disrupted in habitually sedentary, regularly menstruating, exercising women in our experiments.[18]

DiPietro and Stachenfeld also blatantly misrepresent the results of a study[50] in support of their claim that ''energy availability does not always seem to be a mechanism involved in changes in reproductive function among athletes.'' It should be noted first that no one has ever asserted that all menstrual disorders in athletes are caused by low energy availability. Amenorrhoea is a symptom of many serious diseases and of pregnancy, which is why amenorrhoea in athletes should always be differentially diagnosed promptly. In supporting their claim, however, DiPietro and Stachenfeld write about the high incidence of subclinical menstrual disorders found in regularly menstruating, recreational runners that ''only anovulatory women in this study had significantly attenuated energy availability.'' In fact, table 7 in that paper clearly reports that energy availability was significantly and substantially reduced during ovulatory (–25%) and luteally (–16%) deficient as well as anovulatory (–35%) cycles.

Finally, DiPietro and Stachenfeld's sixth allegation is also false. They write that ''the data on the female athlete triad thus far provide little evidence of either a triad or its specificity to athletes.'' The data to which they refer are from epidemiological observational studies and not controlled experiments. DiPietro and Stachenfeld's favourite epidemiological study is a recent one that, contrary to previous studies, found the incidence of the triad to be low and no higher in athletes than in non-athletes.[52] The methodological errors that biased the results of that study have been described at greater length elsewhere.[53 54] Briefly, methods that assess eating disorders instead of measuring energy availability, that measure no hormones, that systematically underestimate and do not differentially diagnose menstrual disorders, and that do not look for declining bone mineral density will yield erroneous data on the incidence of the female athlete triad regardless of how many subjects participate. The female athlete triad is not easy or inexpensive to diagnose, and its epidemiology cannot be determined by survey methods.

DiPietro and Stachenfeld object that by the current understanding of the female athlete triad, ''a single athlete need not have progressed to a clinically diagnosed eating disorder, amenorrhoea, or osteoporosis to be classified as having met the criteria for the female athlete triad.'' In fact, the 1997 ACSM Position Stand did not require the simultaneous presentation of all three pathological extremes to warrant intervention either. It warned that ''Alone or in combination, Female Athlete Triad disorders can decrease physical performance and cause morbidity and mortality'' and advised that ''Sports medicine professionals need to be aware of the interrelated pathogenesis and the varied presentation of components of the Triad. They should be able to recognise, diagnose, and treat or refer women with any one component of the Triad.'' DiPietro and Stachenfeld go on to complain that ''Unfortunately, this new definition of the triad may have extended the classification so broadly that almost any woman (athlete or not) could be considered 'at risk'.'' Indeed so, because ''risk'' in this context refers not to a mere epidemiological statistic on the incidence of pathologies, but rather to experimentally proven physiological mechanisms linking nutrition to reproductive function and bone turnover, which do, indeed, operate in everyone. The female athlete triad was not named because its pathologies occur only in female athletes. It was named because the occurrence of those pathologies in female athletes was alarmingly common and surprisingly unexpected owing to the known stimulation of bone by physical activity.

In summary, DiPietro and Stachenfeld's allegations are all false.

A B Loucks
Ohio University, Athens, Ohio, USA; loucks@ohio.edu

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Refutation of the myth of the female athlete triad

We are pleased to respond to *Refutation of ''the myth of the female athlete triad''* by Loucks; however, to respond in a point-by-point manner to each and every issue of contention would prove quite tiresome, and, more than likely, futile. Therefore, our response will focus on the more general issues of science and language, with particular attention to the translation (ie, application) of laboratory findings into practice and then into policy intended to affect collective behaviours. In doing so, we wish to remind the reader that there are guidelines governing the delicate balance between science and practice. We have described these guidelines previously with regard to the triad,[1] and wish to reiterate that they were developed to prevent practitioners, policy makers and regulators from reacting either too hastily to incomplete science or too slowly to sound science. We continue to maintain that the science pertaining to the female athlete triad is less than complete. Therefore, any attempt to influence practice or policy with regard to the triad should be made with extreme caution, as these efforts may be misguided at this time.

Loucks *et al*[2–5] have made a landmark contribution to women's health by identifying a mechanism (low energy availability, independent of exercise stress) by which exercise disrupts leutinising hormone pulsatility. That this mechanism was identified using the strictest of experimental methods lends substantial internal validity to their findings. Moreover, menstrual function changes were then reversed when energy availability was returned to match energy expenditure, thereby giving even more credibility to the data (as well as to our argument). Whether one feels comfortable in generalising these short-term laboratory-based data from non-athletes and primates to the general population of female athletes who train and compete over many years under real-life conditions is another matter altogether. Dr Loucks *et al*[6 7] may have little use for observational studies; however, Torstveist and Sundgot-Borgen have contributed the best population-based data to date on the issue of the triad.[8 9] At the very least, they provide prevalence estimates of the individual triad components and in toto that were collected from real athletes and a representative control group, rather than from small, select laboratory samples of non-athletes. But here lies the scientific quandary: the experimental data provide us with a necessary biological mechanism, yet the epidemiological data provide little evidence of the pathophysiological relevance of this mechanism to health and function among the population. Which of these two components of science is more important to practice and policy?

In any case, as important as the identification of a biologically plausible mechanism is to the aetiological relationship between exercise and menstrual function changes, one proposed mechanism alone is hardly sufficient to predict the purported risk of triad-related pathophysiology over the lifespan among women. In fact, a constellation of host and environmental factors will also influence one's susceptibility or resistance to menstrual function changes and bone loss, probably even in the presence of low energy availability. If this were not the case, most currently competitive athletes would be sitting on the sidelines with stress fractures, and the infertility clinics currently would be overflowing with former athletes. Needless to say, neither of these scenarios is occurring. Presumably, we will need to wait 30–40 more years to see if these same former athletes are filling the nursing homes with hip fractures because of low bone accrual during adolescence. This would be the value of large-scale epidemiological research that followed up a cohort of female athletes (and controls) through their competitive years and beyond into middle and older age to study longitudinally the influence of low energy availability in adolescence and young adulthood on infertility and osteoporosis later on. At the very least, former National Collegiate Athletic Association (NCAA) athletes (those first awardees of athletic scholarships from 1975 to 1980) currently can be assessed cross-sectionally in middle age and compared with their non-athletic peers. Oddly, we have not encountered any such data, presumably because it is expensive to perform these studies properly with objective biomarkers and measures of energy availability, endocrine function and bone resorption. On the other hand, if the triad is really an issue of such high clinical and public health significance as implied, one would think that the National Institutes of Health, the Centers for Disease Control and Prevention, the World Health Organization or at least the NCAA itself would be willing to fund such a venture.

We did not contribute to the writing of the female athlete triad position stand, or to the